1985年7月7日

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CURRENT CLINICAL TOPICS IN INFECTIOUS DISEASES

JACK S. REMINGTON, M.D.

重庆医学院第一医院 图书室

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McGraw-Hill Book Company

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Johannesburg Lisbon London Madrid Mexico Montreal New Delhi Panama Paris
San Juan São Paulo Singapore Sydney Tokyo Toronto

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"The Library of Congress has cataloged this serial publication as follows:"

Current clinical topics in infectious diseases. 1-
New York, McGraw-Hill Book Co., c1980-
v. ill. 25cm
Annual.
Key title: Current clinical topics in infectious diseases, ISSN 0195-3842.
I. Communicative diseases—Periodicals.
DNLM: 1. Communicable Diseases—Periodicals. W 1 CU786T
RC111.C87 616.9'05 80-643590
ISBN 0-07-051854-8

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1 2 3 4 5 6 7 8 9 0 HALHAL 8 9 8 7 6 5 4

ISBN 0-07-051854-8

This book was set in Electra by TriStar Graphics. The editors were Robert P. McGraw and Maggie Schwarz; the production supervisor was Avé McCracken.
Halliday Lithograph Corporation was printer and binder.

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Prostatitis syndromes

EDWIN M. MEARES, JR.

INTRODUCTION

In their practice, physicians frequently encounter male patients who seem to have prostatic inflammation, or prostatitis. Unfortunately, "prostatitis" remains an often confusing and controversial entity, both to physicians and to patients. Indeed, clinicians and investigators often use nonstandard and imprecise methods of diagnosis that lump together prostatitis syndromes whose cause, significance, sequelae, and proper clinical management vary considerably.

Therapies for prostatitis advocated by some clinicians as highly effective often seem ineffective to others. Why? Because clinicians attempt to treat patients who have dissimilar types of prostatic inflammation with a single form of therapy. An analogy can be made to expected outcomes in the treatment of pneumonia. Few clinicians would expect therapy using penicillin to be equally effective in the management of cases of pneumonia caused by different agents—the pneumococcus, mycoplasmas, or gram-negative bacteria. In a similar manner one would not expect therapy using penicillin to be efficacious in the management of hypersensitivity pneumonitis. Why, then, should clinicians expect uniform results for a particular therapy for prostatitis when they apply such therapy to patients who have prostatitis of variable cause? The answer is clear: specific etiologic diagnosis is essential and is the key to the successful treatment of prostatitis.

Although many questions remain unanswered concerning certain aspects of prostatitis, especially nonbacterial prostatitis, the work of several investigators during recent years has clarified important basic features of inflammations of the prostate and established clear directions for future investigations. In this article these findings will be reviewed and areas that require additional study will be emphasized.

TYPES OF PROSTATITIS

As shown in Table 1, several varieties of prostatitis, or prostatitis syndromes, are recognized. Because appropriate clinical management varies considerably accord-

ing to the underlying cause of the prostatitis, diagnostic specificity is important. In 1978, Drach, Fair, Meares, and Stamey (1) proposed a new classification of common types of prostatitis syndromes: acute and chronic bacterial prostatitis, nonbacterial prostatitis, and prostatodynia (Table 1).

Bacterial prostatitis usually is associated with bacteriuria, and persistence of the pathogen in the prostate in cases of chronic bacterial prostatitis characteristically leads to recurrent bacteriuria in untreated patients. In contrast, nonbacterial prostatitis and prostatodynia, syndromes of uncertain cause, are not associated with urinary tract infection. Similar to patients with bacterial prostatitis, patients with nonbacterial prostatitis have excessive numbers of inflammatory cells in their prostatic secretions, clearly suggesting an inflammatory condition; in contrast, patients with prostatodynia typically have normal prostatic expressates.

ETIOLOGY AND PATHOGENESIS

The pathogens responsible for bacterial prostatitis are similar in type and prevalence to those that cause urinary tract infection: strains of *Escherichia coli* clearly predominate, although infections due to species of *Proteus, Klebsiella, Enterobacter, Pseudomonas, Serratia,* and other, less common gram-negative bacteria are found occasionally (2). Most prostatic infections are caused by a single pathogen, although, at times, mixed infections with two or more strains or types of bacteria are found.

Table 1 Classification of prostatitis

Common types
Acute bacterial prostatitis
Chronic bacterial prostatitis
Chronic bacterial prostatitis with infected calculi
Nonbacterial prostatitis
Prostatodynia
Uncommon types
Gonococcal prostatitis
Tuberculous prostatitis
Parasitic prostatitis
Mycotic prostatitis
Nonspecific granulomatous prostatitis
1 Noneosinophilic variety
2 Eosinophilic variety
Suspected but unproven types
Prostatitis due to ureaplasmas (mycoplasmas)
Prostatitis due to *Chlamydia trachomatis*
Prostatitis due to viruses

Opinions still vary concerning the role played by gram-positive bacteria as causative agents in prostatitis. Most agree that enterococci cause chronic bacterial prostatitis and its associated recurrent bacteriuria; however, whether other gram-positive organisms, such as coagulase-negative staphylococci, streptococci, micrococci, and diphtheroids, are important pathogens in prostatitis is questioned. Drach (3,4) believes that gram-positive bacteria are the most common causative agents in prostatitis. However, since these normal "skin inhabitants" typically colonize the anterior urethra in men, they generally are considered commensals, not pathogens (5). Moreover, longitudinal studies of men who have only gram-positive bacteria, other than enterococci, on localization cultures seldom show reproducible patterns that would prove the existence of prostatitis or a tendency for these organisms to cause urinary tract infection—a hallmark of chronic prostatitis caused by gram-negative bacteria (5). The observations of several investigators indicate that chronic prostatitis due to gram-positive bacteria other than enterococci is uncommon and insignificant (5–10).

The specific cause of most cases of nonacute prostatitis is unknown. In addition, important aspects concerning the route of infection and pathogenesis of bacterial prostatitis remain uncertain. Possible routes of infection include (1) ascending urethral infection; (2) reflux of infected urine into prostatic ducts that empty into the posterior urethra; (3) invasion by rectal bacteria, by direct extension or lymphogenous spread; and (4) hematogenous infection.

Infectious types of prostatitis may be sexually transmitted. Gonococcal and nongonococcal urethritis develop in men as ascending infection following inoculation of the urethral meatus by vaginal organisms during sexual intercourse. Gonococcal prostatitis occurs only in men with prior gonococcal urethritis. Male sexual partners of women who have abnormal vaginal cultures due to coliform bacteria at times have simultaneously positive urethral cultures for the same coliforms (11). Generally, these men are asymptomatic and their cultures revert to normal spontaneously. On occasion, however, the same pathogenic bacteria are found in prostatic fluid cultures of men with chronic bacterial prostatitis and in vaginal cultures of their sexual partners, implying transmission by sexual means (11,12). The frequency and importance of this mode of infection in cases of prostatitis need substantiation by additional study.

The likelihood that reflux of infected urine into prostatic ducts is an important mode of infection in cases of prostatitis is enhanced by the recent observation that many prostatic calculi on crystallographic analysis contain constituents common to urine but foreign to prostatic secretions (13). This implies that at times urine must enter the prostatic ducts, presumably by reflux.

Many cases of bacterial prostatitis seem to occur as a consequence of periurethral infection associated with indwelling urethral catheterization.

METHODS OF DIAGNOSIS

General comments

Too often the diagnosis of prostatitis is made by the clinician without substantiation. Often, the medical history and physical findings are not helpful in confirming

a diagnosis of prostatitis. For instance, although acute bacterial prostatitis usually is recognized easily because its clinical manifestations are quite characteristic, the clinical features of chronic prostatitis are highly variable and inexact. Indeed, many of the signs, symptoms, and physical findings in cases of chronic bacterial prostatitis, nonbacterial prostatitis, and prostatodynia often are indistinguishable. Similarly, x-ray studies and cystourethroscopy do not specifically confirm the diagnosis of prostatitis.

Histological examination of prostatic tissue generally is required to establish the diagnosis of unusual types of prostatitis, such as granulomatous prostatitis. Because conditions other than bacterial infection produce histological changes of tissue reaction to injury similar to those seen in chronic bacterial prostatitis, findings in prostatic tissue specimens are not specific for chronic bacterial prostatitis. Recently, Kohnen and Drach (14) studied a series of 162 consecutive cases of surgically resected, hyperplastic prostates and found an incidence of inflammation of 98.1 percent. Six distinct morphological patterns of inflammation were described. No significant differences among these patterns were identified among groups of cases with and without cultural evidence of bacterial prostatic infection. Thus, prostatic biopsy is seldom indicated in the diagnosis and management of the usual case of prostatitis. Tissue culture of specimens obtained by prostatic biopsy are not recommended in the diagnosis of chronic bacterial prostatitis; since the infection of the gland typically is focal, sampling errors are significant. Furthermore, these tissue specimens are difficult to culture quantitatively and are easily contaminated during procurement.

Examination of expressed prostatic secretions

Microscopic examination of the expressed prostatic secretions is an important aid in diagnosis but can be misleading. False impressions regarding leukocytosis in these secretions occur in urethral disease such as urethritis, strictures, condylomata, and diverticula, as well as in noninfectious conditions of the prostate, such as uninfected prostatic calculi. Moreover, the leukocyte count in prostatic fluid often rises significantly in healthy men for several hours following sexual intercourse and ejaculation (15).

To localize the site of inflammatory response to the urethra or prostate, the clinician always should compare the microscopic appearance of the expressed prostatic secretions (expressate) to smears of the spun sediment of the first-voided 10 ml of urine (urethral specimen) and the midstream urine (bladder specimen). Continuing controversy prevails concerning what constitutes an abnormal number of leukocytes in prostatic secretions. Most clinicians agree that >20 white blood cells per high-power field is excessive; some prefer the criterion of >15 white blood cells per high-power field; others believe that >10 white blood cells per high-power field represents leukocytosis (1,5). The work of Blacklock (16) and recent studies by Anderson and Weller (17) and Schaeffer et al. (18) convincingly indicate that prostatic fluid normally contains no more than 10 white blood cells per high-power field.

The most convincing sign of prostatic inflammation is the finding of both

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leukocytosis and excessive numbers of lipid-laden macrophages (oval fat bodies) in the prostatic expressate (5,16). These macrophages containing fat are seldom noticed in the prostatic secretions of healthy men, are increased about eightfold in men with nonbacterial prostatitis, and often are exceedingly prominent in secretions of men who have bacterial prostatitis. Further, macrophages containing fat are not found in exudates arising from the urethra.

Examination of semen

Isolated analysis or culture of the ejaculate can be even more misleading than isolated examination of the prostatic expressate. Not only is urethral contamination a concern, but the semen is composed of fluids from various sites and organs. Moreover, cytological examination of the semen is complicated by the difficulty of distinguishing immature sperm from leukocytes. Mobley is an advocate of semen cultures for diagnosis of bacterial prostatitis and has based some of his clinical studies, at least in part, upon this method (19–21). However, unless one obtains a quantitative culture from the urethra and bladder urine immediately before obtaining the semen specimen for quantitative culture, proper interpretation of the culture results is impossible. Indeed, urethral organisms of nonprostatic origin can easily contaminate the semen as it traverses the urethra during procurement.

Measurements of immune response

In 1963, Chondirker and Tomasi (22) reported the presence of IgG and IgA in normal human prostatic fluid in mean concentrations of 157 mg/dl and 26 mg/dl, respectively. Subsequently, Gray et al. (23) used methods of immunodiffusion and immunoelectrophoresis and compared the immunoglobulin concentrations in expressed prostatic secretions of 33 healthy men with those of 48 men with prostatitis. These investigators established the normal levels of IgA, IgG, and IgM and further noted that the IgA present in all controls and patients was entirely secretory IgA. Among patients with "unresolved" prostatitis, they observed significantly increased levels of prostatic fluid immunoglobulin: IgA (sevenfold), IgG (threefold), and IgM (2.5–fold), compared with normal men. Among patients whose prostatitis was thought to have been resolved, they noted a return of the IgG level to normal but persistent elevation of the IgA and IgM levels at twice normal. Although these investigators believe that the elevated levels of secretory IgA in the prostatic secretions of the patients with prostatitis denote the presence of a foreign antigen, they did not study the cause of the prostatitis or the antigens that may have been responsible.

When studied by a method of O-specific direct bacterial agglutination, 18 (82 percent) of 22 men with chronic prostatitis due to strains of *E. coli* had elevated serum antibody titers ($\geq$1:320) against the bacteria in their prostates, whereas men who had only urethritis due to *E. coli* and normal men had uniformly low titers ($\leq$1:160), respectively, against the *E. coli* in their urethras and feces (24). This technique was used further to show a return to normal of previously elevated serum antibody titers in men after cure of chronic prostatitis due to *E. coli*,

whereas serum titers remained elevated in men who were treatment failures (25). Although this method is thought to measure mainly IgM, an immunologic response was demonstrated in serum against prostatic pathogens in patients with chronic bacterial prostatitis.

Thomas et al. (26) used a technique of direct immunofluorescence and observed a positive test for antibody-coated bacteria in the urine from four of five men with prostatitis. Jones (27) not only confirmed this finding but demonstrated specificity of the antibody for the infecting *E. coli* in a patient with chronic prostatitis. Subsequently, various investigators studied immunoglobulins in the ejaculate of patients with prostatitis and reported increased levels, particularly of IgA (28–30). Because the semen is an admixture of fluids from various sites, not only is the specificity of this unclear, but increased levels of IgA and postitive antibody-coated bacteria tests have been found in the prostatic fluid and urine in patients with benign prostatic hyperplasia, prostatic carcinoma, bladder tumors, and bladder stones without evidence of accompanying prostatitis (29,31).

In summary, these studies have demonstrated elevated levels of total immunoglobulin and high levels of antigen-specific antibody in the prostatic secretions and elevated levels of antigen-specific antibody in the serum against the prostatic pathogens in cases of chronic bacterial prostatitis.

More recently, Shortliffe, Wehner, and Stamey (32) used a solid-phase radioimmunoassay to follow longitudinally the immune response in serum and prostatic secretions of men with acute and chronic bacterial prostatitis. These studies demonstrated a distinct local antibody response (mainly secretory IgA) in prostatic fluid that was independent of the serum response and that was antigen-specific for the infecting pathogen. More recently, Fowler and associates (33,34) also used a solid-phase radioimmunoassay to study the immune response of the prostate to bacteriuria and bacterial prostatitis. Their data suggest that bacteriuria is associated with increased secretion of IgA in the prostatic fluid independent of prostatic infection and independent of systemic immune response. These investigators believe the prostate may be colonized routinely during episodes of urinary tract infection but that symptomatic or chronic infections are prevented by the secretion of antigen-specific IgA.

From these preliminary studies it is clear that the prostate elaborates an immune response in instances of bacterial prostatitis. Further elucidation of this response may provide important clues concerning the diagnosis, management, and possible prevention of bacterial prostatitis.

Bacteriologic cultures for localization of infection

The most accurate method of distinguishing between bacterial prostatitis and nonbacterial forms of prostatitis and of establishing the diagnosis of chronic bacterial prostatitis is the performance of essentially simultaneous, quantitative bacteriologic cultures of the urethra, bladder urine, and expressed prostatic secretions. Details regarding the collection of specimens, methods of culture, and interpretation of results have been reported previously (35). The important features of this technique will be summarized here.

Collection of specimens The voided urine and expressed prostatic secretions are partitioned into segments: (1) the first-voided 10 ml of urine (VB_1, voided bladder 1)—urethral sample; (2) the midstream aliquot (VB_2)—bladder sample; (3) the prostatic secretions expressed by prostatic massage (EPS, expressed prostatic secretions)—prostatic fluid sample; and (4) the first-voided 10 ml of urine immediately after prostatic massage (VB_3)—prostatic fluid sample (Figure 1, Table 2).

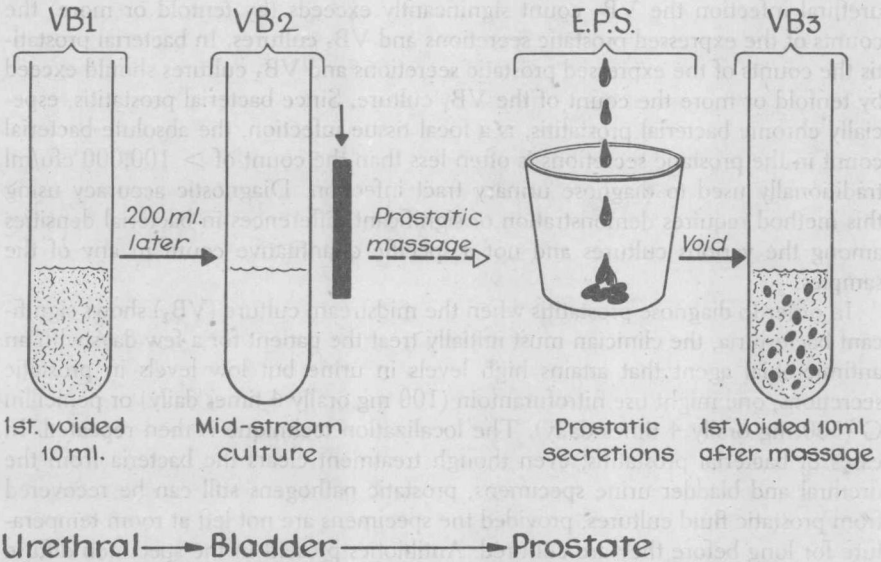

Figure 1 Segmented cultures of the lower urinary tract in the male. EPS = expressed prostatic secretions. *From Meares EM Jr, Stamey TA (35).*

Table 2 Technique of specimen collection in performing diagnostic localization cultures for prostatitis

1 Patient should have a full bladder and desire to void.
2 Prepare skin of glans in uncircumcised man (generally unnecessary in circumcised man).
3 Maintain full retraction of foreskin throughout all collections.
4 Collect first 10 ml voided directly into sterile tube (urethral sample).
5 Take midstream specimen after patient voids about 200 ml (bladder sample).
6 Patient stops voiding and bends forward.
7 Collect drops of prostatic secretions directly into sterile container during prostatic massage (prostatic sample).
8 Patient then voids immediately—collect first 10 ml (prostatic sample).
9 Refrigerate all samples immediately until cultures are performed.

Quantitative culture methods The specimens are refrigerated immediately until quantitative culture techniques are performed. One-tenth milliliter of each specimen is surface-streaked onto both blood agar and MacConkey agar. After incubation for 24 to 48 h, bacterial colonies are counted and multiplied by 10 to give the quantitative count of bacterial colony–forming units per milliliter. Standard bacteriologic methods of organism identification are used.

Diagnostic interpretation When the bladder urine (VB_2) is sterile or nearly so, pathogenic bacteria generally can be localized to the urethra or prostate by a comparison of the bacterial counts of the urethral and prostatic specimens. In urethral infection the VB_1 count significantly exceeds (by tenfold or more) the counts of the expressed prostatic secretions and VB_3 cultures. In bacterial prostatitis the counts of the expressed prostatic secretions and VB_3 cultures should exceed by tenfold or more the count of the VB_1 culture. Since bacterial prostatitis, especially chronic bacterial prostatitis, is a focal tissue infection, the absolute bacterial count in the prostatic secretions is often less than the count of $>$ 100,000 cfu/ml traditionally used to diagnose urinary tract infection. Diagnostic accuracy using this method requires demonstration of significant differences in bacterial densities among the various cultures and not a specific quantitative count in any of the samples.

In order to diagnose prostatitis when the midstream culture (VB_2) shows significant bacteriuria, the clinician must initially treat the patient for a few days with an antimicrobial agent that attains high levels in urine but low levels in prostatic secretions; one might use nitrofurantoin (100 mg orally 4 times daily) or penicillin G (500 mg orally 4 times daily). The localization technique is then repeated. In cases of bacterial prostatitis, even though treatment clears the bacteria from the urethral and bladder urine specimens, prostatic pathogens still can be recovered from prostatic fluid cultures, provided the specimens are not left at room temperature for long before they are cultured. Antibiotics present in the specimen diffuse into the agar, permitting bacteria to grow on the surface of the agar plate. Indeed, positive prostatic fluid cultures with accompanying sterile urethral and bladder urine cultures provide the best documentation of bacterial prostatitis. Selected clinical examples of the usefulness of these techniques for diagnosis of chronic bacterial prostatitis are shown in Table 3.

ACUTE BACTERIAL PROSTATITIS

Clinical features

Acute bacterial prostatitis is characterized by the sudden onset of chills and fever, perineal and low back pain, urinary frequency and urgency, nocturia, dysuria, generalized malaise and prostration, myalgia and arthralgia, and varying degrees of symptoms of bladder outlet obstruction. Rectal examination discloses a very tender, swollen prostate that is partly or totally quite firm and warm to touch. Although prostatic massage will produce grossly purulent prostatic secretions from which the pathogen can be isolated in large numbers on culture, massage should

Table 3 Duplicate set of localization cultures in chronic bacterial prostatitis

				Colonies per milliliter		
Patient	*Antibiotic*	*VB_1*	*VB_2*	*EPS*	*VB_3*	*Organism*
1	Yes	20	0	6,000	600	*E. coli*
1	Yes	530	200	40,000	2,000	*E. coli*
2	No	60	0	1,000	20	*E. coli*
2	No	640	40	100,000	1,200	*E. coli*
3	Yes	0	0	1,000	0	*Enterococcus*
3	Yes	20	0	4,000	20	*Enterococcus*
4	No	1,000	300	100,000	10,000	*E. coli*
4	Yes	100	0	10,000	1,000	*E. coli*
5	Yes	0	0	7,000	200	*P. mirabilis*
5	Yes	0	0	10,000	600	*P. mirabilis*
6	Yes	250	20	5,000	400	*K. pneumoniae*
6	Yes	0	0	10,000	1,300	*K. pneumoniae*
7	Yes	50	0	7,000	500	*E. coli*
		20	10	10,000	1,000	*P. aeruginosa*
7	Yes	0	0	5,000	400	*E. coli*
		20	0	8,000	1,000	*P. aeruginosa*
8	No	1,000	100	100,000	10,000	*E. coli*
8	Yes	30	10	10,000	2,000	*E. coli*
9	Yes	50	10	10,000	750	*E. coli*
9	Yes	0	0	15,000	1,000	*E. coli*
10	Yes	0	0	600	200	*E. coli*
		0	0	500	30	*P. mirabilis*
10	Yes	0	0	400	100	*E. coli*
		0	0	600	50	*P. mirabilis*

NOTE: VB_1 = first-voided 10 ml of urine (urethral culture); VB_2 = midstream urine (bladder culture); EPS = expressed prostatic secretions (prostatic culture); VB_3 = first-voided 10 ml immediately after prostatic massage (prostatic culture).

be avoided because it is painful for the patient and may cause bacteremia. Since bacteriuria usually accompanies acute prostatitis, the causative agent generally can be identified by culture of the voided urine.

Treatment

Hospitalization may be required. General supportive measures, i.e., bed rest, hydration, medicine for fever and pain, and stool softeners, are indicated. If significant urinary retention requires bladder drainage, placement of a punch suprapubic tube under local anesthesia is preferred to use of an indwelling urethral catheter.

The intense and diffuse inflammation of acute prostatitis apparently allows

many drugs that normally are excluded to diffuse readily into prostatic secretions. However, we still prefer to initiate therapy using trimethoprim-sulfamethoxazole (T-S), 160 mg trimethoprim and 800 mg sulfamethoxazole orally twice daily, until the results of the culture and sensitivity testing are known. If the pathogen is susceptible, and if the clinical response is favorable, we continue therapy using this dosage for at least 30 days. Our goal in using prolonged therapy is to prevent the development of chronic bacterial prostatitis. As an alternative, initial therapy using gentamicin or tobramycin, 3 to 5 mg/kg per day (divided into three equal intravenous or intramuscular doses) plus 2 g ampicillin given intravenously every 6 h, is recommended until the results of the culture and susceptibility tests are known. A suitable oral antimicrobial agent can be substituted after about 1 week and continued for at least 30 days in full dosage.

CHRONIC BACTERIAL PROSTATITIS

Clinical features

Even though its clinical manifestations are quite variable, chronic bacterial prostatitis is a common cause of relapsing urinary tract infection in men. Often, history of a prior bout of acute prostatitis is absent. Some men are diagnosed only because asymptomatic bacteriuria is found incidentally; however, most complain of variable degrees of irritative voiding dysfunction and pain perceived in various sites within the distribution of the pelvis and genitalia. Some experience postejaculatory discomfort, with or without hemospermia. Chills or fever are unusual. On rectal examination there are no characteristic or specific findings; the same is true for cystoscopy and urography.

The hallmark of chronic bacterial prostatitis is the occurrence of relapsing urinary tract infection caused by the same pathogen. The organism persists unaltered in prostatic secretions during therapy with most antimicrobial agents because most of these drugs diffuse poorly from plasma into prostatic fluid. The urine may be sterilized and symptoms controlled during treatment; however, discontinuation of therapy eventually leads to reinfection of the urine and recurrence of symptoms. Diagnosis is confirmed by performance of bacteriologic cultures that localize the pathogen to the prostatic secretions (Table 3).

Infected prostatic calculi

Prostatic calculi are common findings in the middle-aged and elderly. Fox (36) reviewed pelvic x-ray films of 3510 men and observed an incidence of radiopaque prostatic calculi of 1.9 percent among men under age 25 years compared with 20 percent among men over age 65 years. Detailed examination of surgical and autopsy specimens, however, indicates that tiny prostatic stones, often invisible on x-ray films of the pelvis, occur in almost every adult prostate.

Typically, prostatic stones are minute but occur in small clusters. Multiple large stones are seen most often in men who have chronic bacterial infection of the prostate. Prostatic calculi usually cause no symptoms or apparent harm. However,

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